REMARKS

Reconsideration is requested.

Claims 1-2, 10 and 43-44 are pending. Support for the amended claims can be found throughout the specification. No new matter has been added.

The Section 102 rejection of claims 1 and 10 over Kassis (Clinical Cancer Research, August 1999, vol 5, pp 2251-2260) is obviated by the above amendments. Withdrawal of the rejection is requested in view of the following further comments.

The claims define a method which requires a monoclonal antibody which specifically recognizes 1175-tyrosine phosphorylated KDR/Flk-1 (referred to as "anti-PY1175 antibody").

Kassis et al. neither disclose nor suggest that KDR/Flk-1 signal transduction can be inhibited by contacting endothelial cells with the anti-PY1175 antibody as disclosed and presently claimed. The claims are therefore submitted to be patentable over the cited art. Withdrawal of the Section 102 rejection is requested.

To the extent not obviated by the above amendments, the Section 112, first paragraph "enablement", rejection of claims 2, 10-13 and 42 is traversed. Reconsideration and withdrawal of the rejection are requested in view of the following further comments and the attached.

The presently claimed invention relates to a method for inhibiting KDR/Flk-1 signal transduction in endothelial cells and a method for inhibiting cell growth of endothelial cells, which comprises contacting endothelial cells with the anti-PY1175 antibody. The attached Declaration demonstrates that the cell growth of endothelial cells is inhibited by contacting vascular cells with the anti-PY1175 antibody. That is, the

Declaration demonstrates that the cell growth of endothelia cells accelerated by VEGF can be inhibited by contacting human microvascular endothelial cells with the anti-1175-tyrosine phosphorylated KDR/Flk-1 monoclonal antibody prepared in Example 6 of the present specification. Accordingly, since the anti-PY1175 antibody of the present invention can inhibit cell growth of endothelial cells, claims 2 and 10, for example, are submitted to be supported by an enabling disclosure.

Furthermore, Example 5 in the present specification shows that incorporation of BrdU into endothelial cells stimulated by VEGF is inhibited by the anti-PY1175 antibody of the present invention. That is, it is demonstrated that, as a result that KDR/Flk-1 signal transduction in endothelial cells is inhibited by the anti-PY1175 antibody of the present invention, the BrdU incorporation activity stimulated by VEGF is inhibited. Accordingly, since the anti-PY1175 antibody inhibits KDR/Flk-1 signal transduction in endothelial cells, the claims, such as claims 1 and 10, are believed to be supported by an enabling disclosure.

Withdrawal of the Section 112, first paragraph, rejection of claims 2, 10-13 and 42 is requested.

The claims are submitted to be in condition for allowance and a Notice to that effect is requested. The Examiner is requested to contact the undersigned in the event anything further is required.

Respectfully submitted,

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